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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/526,741	11/14/2005	Hiroyuki Aburatani	392.1001	4248

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EXAMINER	
REDDIG, PETER J	

ART UNIT	PAPER NUMBER
1642	

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09/01/2009	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/526,741	ABURATANI ET AL.	
	Examiner	Art Unit	
	PETER J. REDDIG	1642	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *24 March 2008*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *9, 23-29* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *9, 23-29* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *8/10/2009*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on August 10, 2009 has been entered. Claims 9 and 23-29 are currently being examined.

Rejections Maintained

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

The factual inquiries set forth in *Graham* **v.** *John Deere Co.*, 383 U.S. 1, 148 USPQ 459 (1966), that are applied for establishing a background for determining obviousness under 35 U.S.C. 103(a) are summarized as follows:

1. Determining the scope and contents of the prior art.
2. Ascertaining the differences between the prior art and the claims at issue.
3. Resolving the level of ordinary skill in the pertinent art.
4. Considering objective evidence present in the application indicating obviousness or nonobviousness.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any

evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

2. Claims 9 and 23-29 are rejected under 35 U.S.C. 103(a) as being unpatentable over Lage et al. (Virchows Arch 2001 438:567-573, IDS), in view of Steplewski et al. (Proc. Natl. Acad. Sci. USA, 1988 85: 4852-4856), further in view of Dillman et al.(Annals of Internal Medicine 1989, 111:592-603), further in view of Mast et al. (Biochem. J. 1997, 327: 577-583), and further in view of Midorikawa (Proc. Amer. Assoc. Can. Res. March 2002, 43:11 Abstract #53). for the reasons previously set forth in the Office Action of July 9, 2008, section 2-pages 3-5, which are set forth below.

Lage et al. teach the production of a monoclonal antibody to GPC3 using an oligopeptide of amino acids 537-556 of human GPC3, see Materials and Methods. Lage et al. use the standard art technique of fusing spleen cells from immunized mice with myeloma cells to generate hybridomas for the production of the monoclonal antibody, which leads to recombination of the fused cellular genomes, thus the monoclonal antibodies are recombinant antibodies. Lage et al. teach that the glycosyl-phosphatidylinositol anchor GPC3 protein is expressed in hepatocellular carcinomas, decreasing in expression in tumor grade, see Abstract, table 1, Fig. 2-4.

Lage et al. does not teach that the antibody has any cytotoxic activity in vitro against the cell line HepG2 in the presence of complement or peripheral blood mononuclear cells or a humanized form of the antibody.

Steplewski et al. teach that mouse monoclonal antibodies are humanized to overcome the problem of short half-life and immunogenicity of murine monoclonal antibodies in humans, see page 4852, first paragraph. Steplewski et al. teach the generation of humanized mouse monoclonal anti-bodies using Cγ1, Cγ2, Cγ3, and Cγ4 human heavy chains and human Cκ light chains, see Materials and Methods. Steplewski et al. that these humanized antibodies can mediate antibody dependent cell mediated cytotoxicity, ADCC, *in vitro* in the presence of peripheral blood monocytes, see Abstract, Materials and Methods, and Fig.4.

Dillman teaches that IgG1, IgG2, IgG3, and IgG4 humanized mouse monoclonal antibodies mediate complement mediated cytotoxicity, see p. 593, 1st col.

Mast et al. teach that GPC3 is expressed on the surface of HepG2 cells, see Title, Abstract, and Introduction.

Midorikawa et al. teach that GPC3 protein is found in elevated levels in HepG2 cells and 22 of 52 hepatocellular carcinomas examined.

It would be *prime facia* obvious to one of skill in the art at the time the invention was made to humanize the monoclonal antibody of Lage et al. using the methods of Steplewski et al. to make humanized monoclonal antibodies that have Cγ1, Cγ2, Cγ3, or Cγ4 human heavy chains and human Cκ light chains that have cytotoxic activity in the presence of complement or peripheral blood mononuclear cell as Steplewski et al. teach that humanization of antibodies is done to overcome the problems of using mouse monoclonal antibodies in human therapy. Additional Steplewski et al. teach that these humanized antibodies have cytotoxic activity toward cells expressing the target antigen in the presence of peripheral blood monocytes and Dillman teaches that IgG1, IgG2, IgG3, and IgG4 humanized mouse monoclonal antibodies mediate complement mediated cytotoxicity. One would have been motivated to humanize antibodies the monoclonal antibody of Lage et al. to screen them for potential therapeutic antibodies using HepG2 cells that expressed GPC3 on their surface given that Lage et al. and Midorikawa et al. teach that GPC3 is expressed in hepatocelllular carcinomas, given that HepG2 cells express GPC3 on their cell surface at elevated levels, and given the importance of developing new cancer therapeutics. One of skill in the art would have had a reasonable expectation of success of making a humanized, monoclonal antibody against a peptide consisting of amino acid residues 375-580 of GPC/SEQ ID NO: 4 that has cytotoxic activity in vitro against HepG2 cells in the presence of mononuclear cells or complement given that the monoclonal antibody of Lage et al. binds within amino acid residues 375-580 of GPC3, the methods for humanizing antibodies were well known in the art at the time the invention was made, and the humanized antibodies claimed were known to have to cytotoxic activity in the presence of complement or peripheral blood mononuclear cells.

In the remarks of January 9, 2009 Applicants argue that Lage, at page 570, right-column, lines 9-15 and Fig. 1, discloses that Western-blot analysis of a stomach cancer cell line EPG85-257RNOV using the disclosed antibody Be-F4 revealed the analogous results as the Northern blot analysis of stomach cancer (See Lage, page 570, right-column, line 13-15). On the other hand, Lage discloses the Western-blot analysis and histological staining ofhepatocarcinoma (See Lage, page 571, left-column and right-column text and Figs. 2 and 4) and concludes that "HCC cells constantly showed a decreased staining intensity when compared with the staining signal obtained in noncancerous liver cells from the same section (See Lage, page 571, right column,

line 1-4)" and "[i]n contrast to the non-neoplastic hepatocytes, the cellular p62 content was unambiguously reduced in all malignant cells." (See Lage, Abstract line 11-13).

Applicants argue that it is evident from the disclousre of Lage that GPC3 is not highly expressed in a protein level in hepatocarcinoma cells. Applicants argue that the disclosure of Lage is inconsistent with the disclosure of Midorikawa wherein GPC3 is highly expressed in HepG2 cells and hepatocarcinoma cells, and with the disclosure of Mast that GPC3 is expressed on the surface of HepG2 cells (Applicants note that Mast does not mention the level of expression). Therefore, Applicants respectfully submit that one of skill in the art would not look to combine the teachings of Lage, Midorikawa and Mast because they are inconsistent with each other.

Applicants argue that even if Lage discloses a GPC3 monoclonal antibody obtained using an oligopeptide of amino acids 537-556 of GPC3, one of skill in the art would not combine Lage with Midorikawa and Mast which provide opposite teachings from Lage. Thus, one of skill in the art would not look to combine these references, thus the present invention is not obvious from the inventions disclosed in these references.

Applicants' arguments have been carefully considerd, but have not been found persuasive. Although Lage et al. does not show that GPC3 is highly expressed, it does show that GPC3 is expressed in hepatocellular carcinomas. Thus given the combined teachings of Lage and Midorikawa which both teach that GPC3 is expressed in hepatocellular carcinoma and given that Mast et al. teach that GPC 3 is expressed on the cell surface of HepG2 cells, one of skill in the art would have been motivated with a reasonable expectatin of success of making recombinant humanized monoclonal antibodies that has a cytotoxic activity in vitro against the

cell line HepG2 in the presence of complement or peripheral blood mononuclear cells in view of Steplewski and Dillman who teach how to make humanized monoclonal antibodies that have ADCC or CDC activity in the presence of complement or peripheral blood mononuclear cells.

3. Claims 9 and 23-29 are rejected under 35 U.S.C. 103(a) as being unpatentable over Filmus et al. (US Pat App. Pub. 2005/0233392 A1 May 23, 2002), in view of Steplewski et al. (Proc. Natl. Acad. Sci. USA, 1988 85: 4852-4856), further in view of Dillman et al. (Annals of Internal Medicine 1989, 111:592-603), further in view of Mast et al. (Biochem. J. 1997, 327: 577-583), and further in view of Midorikawa (Proc. Amer. Assoc. Can. Res. March 2002, 43:11 Abstract #53) for the reasons previously set forth in the Office Action of July 9, 20008, section 3-pages 5-7, which are set forth below.

Filmus et al. teach the production of a monoclonal antibody to GPC3 using the last 70 amino acids of human GPC3, see para 0098 and 0107-0109 and claims 7 and 33. Filmus et al. use the standard art technique of fusing spleen cells from immunized mice with myeloma cells to generate hybridomas for the production of the monoclonal antibody, which leads to recombination of the fused cellular genomes, thus the monoclonal antibodies are recombinant antibodies, see para 0098 and 0107-0109. Filmus et al. teach that the monoclonal antibody 1G12 bound strongly to human liver tumor cells, but not normal hepatocytes, see Examples 4 and 5.

Filmus et al does not teach that the antibody has any cytotoxic activity *in vitro* against the cell line HepG2 in the presence of complement or peripheral blood mononuclear cells or a humanized form of the antibody.

Steplewski et al. teach that mouse monoclonal antibodies are humanized to overcome the problem of short half-life and immunogenicity of murine monoclonal antibodies in humans, see page 4852, first paragraph. Steplewski et al. teach the generation of humanized mouse monoclonal anti-bodies using Cγ1, Cγ2, Cγ3, and Cγ4 human heavy chains and human Cκ light chains, see Materials and Methods. Steplewski et al. that these humanized antibodies can mediate antibody dependent cell mediated cytotoxicity, ADCC, *in vitro* in the presence of peripheral blood monocytes, see Abstract, Materials and Methods, and Fig.4.

Dillman teaches that IgG1, IgG2, IgG3, and IgG4 humanized mouse monoclonal antibodies mediate complement mediated cytotoxicity, see p. 593, 1st col.

Mast et al. teach that GPC3 is expressed on the surface of HepG2 cells, see Title, Abstract, and Introduction.

Midorikawa et al. teach that GPC3 protein is found in elevated levels in HepG2 cells and 22 of 52 hepatocellular carcinomas examined.

It would be *prime facia* obvious to one of skill in the art at the time the invention was made to humanize the monoclonal antibody of Filmus et al using the methods of Steplewski et al. to make humanized monoclonal antibodies that have Cγ1, Cγ2, Cγ3, or Cγ4 human heavy chains and human Cκ light chains that have cytotoxic activity in the presence of complement or peripheral blood mononuclear cell as Steplewski et al. teach that humanization of antibodies is done to overcome the problems of using mouse monoclonal antibodies in human therapy. Additional Steplewski et al. teach that these humanized antibodies have cytotoxic activity toward cells expressing the target antigen in the presence of peripheral blood monocytes and Dillman teaches that IgG1, IgG2, IgG3, and IgG4 humanized mouse monoclonal antibodies mediate complement mediated cytotoxicity. One would have been motivated to humanize antibodies the monoclonal antibody of Filmus et al to screen them for potential therapeutic antibodies using HepG2 cells that expressed GPC3 on their surface at elevated levels given that Filmus et al. and Midorikawa et al. teach that GPC3 is expressed in hepatocelllular carcinomas and given the importance of developing new cancer therapeutics. One of skill in the art would have had a reasonable expectation of success of making a humanized, monoclonal antibody against a peptide consisting of amino acid residues 375-580 of GPC/SEQ ID NO: 4 that has cytotoxic activity in vitro against HepG2 cells in the presence of mononuclear cells or complement given that the monoclonal antibody of Filmus et al binds within amino acid residues 375-580 of GPC3, the methods for humanizing antibodies were well known in the art at the time the invention was made, and the humanized antibodies claimed were known to have to cytotoxic activity in the presence of complement or peripheral blood mononuclear cells.

In the remarks of January 9, 2009, Applicants argue that the antibody 1G12, as disclosed in Filmus is also described in International Patent Publication W02007/137170, a copy of which is attached as Appendix A. International Patent Publication W02007/137170 discloses in paragraph [0202] that "The cytotoxic activities of the anti-GPC3 antibodies (1G12, 8H5) with a secondary antibody conjugate were evaluated on the Glypican-3 positive HepG2 and Hep3B cell lines." See International Patent Publication W02007/137170, paragraph [0202]. Applicants argue that the unconjugated antibodies (1G12, 8H5) were not observed to have cytotoxic activity on these cell lines.

Applicants argue that in addition, Applicants submit herewith a Data Sheet, attached as Appendix B (also available from the website

http://www.biomosaics.com/pdfs/B0134R%20%20data%20sheet%202007, pdf), the antibody 1G12 is distributed in a commercially available manner, and a person skilled in the art can easily confirm that the antibody 1G12 does not have cytotoxic activity against hepatocarcinoma cells such as HepG2 cell line.

Applicants argue that in the Office Action, the Examiner alleged inter alia that it was obvious for a person skilled in the art to humanize the monoclonal antibody 1G12 which strongly bind to human hepatocarcinoma cells using the humanizing techniques disclosed in Steplewski to create a humanized antibody having cytotoxicity (see page 7, line 4-15 of the Office Action). However Applicants argue that although Steplewski teaches that the mouse monoclonal antibody CO 17-1A having cytotoxicity may be humanized to overcome the problem of short half-life and immunogenicity, Steplewski fails to disclose that a mouse monoclonal antibody having no cytotoxicity, e.g. 1G12 antibody, may be humanized to impart cytotoxicity.

Applicants argue that the Examiner also alleged that the antibody of Filmus which binds to the amino acids 375-580 of GPC3 was known, and humanized techniques of antibodies as well as humanized antibodies having cytotoxicity were well known in the art, and thus one would prepare a humanized monoclonal antibody against a peptide having the amino acids 375-580 of GPC3 with a reasonable expectation of success (see page 7, line 8-15 of the Office Action). Applicants argue that as discussed above, however, since it was well known in the art, or one could easily understand, that the antibody ofFilmus, 1G12 antibody, does not have cytotoxicity, a person skilled in the art could not have been motivated from Steplewski to prepare a humanized antibody having cytotoxicity by humanizing a mouse monoclonal antibody having no cytotoxicity. More likely, a person skilled in the art would recognize it difficult to prepare a

humanized anti-GPC3 antibody having cytotoxicity based on Filmus that teaches the monoclonal antibody 1 G12 having no cytotoxicity.

Applicants argue that in addition, Filmus discloses that the antibody 1G12 can detect GPC3 in serum (Example 6). Filmus teaches that GPC3 is released from hepatocarcinoma cells. Mast teaches that GPC3 is expressed on the surface of HepG2 cells and Midorikawa teaches that GPC3 level is elevated in HepG2 cells and 22 out of 52 hepatocellular carcinomas, while Filmus teaches that GPC3 expressed in that way is detected in serum. Accordingly, a person skilled in the art seeking to obtain an antibody having cytotoxicity with the aim of achieving treatment of hepatocarcinoma by damaging hepatocarcinoma cells would not look to combine Filmus with Mast and Midorikawa with a reasonable expectation of success, because Filmus discloses that GPC3 is detected in serum and, as discussed, the antibody 1 G12 cannot exert cytotoxicity against HepG2 cells.

Applicants argue that in conclusion, a person skilled in the art could not have conceived of the present invention based on the combination of the cited references.

Applicants arguments have been considered, but have not been found persuasive. Althougt the monoclonal antibodies of Filmus et al. do not have cytoxic activity by themselves, the claims are not drawn to monoclonal antibodies with inherent cytotoxic activity. W0/2007/137170 shows that the 1G12 and 8H5 antibodies can be used to induce cyotoxicity in HepG2 cells in the presence of the appropriate cytoxic stimuli, ie a drug conjugated secondary antibody, see Table 3 of W0/2007/137170. Thus humanized versions of the Filmus et al. monoclonal antibodies with complement or mononuclear cell reactive Fc domains would be expected to have cytotoxic activity towards HepG2 cells in the presence of complement or

peripheral blood mononuclear cells. Although the monoclonal antibody of Steplewski had cytotoxic activity before humanization, given that it was well known in the art that the Fc portion of antibodies impart ADCC and CDC activity to an antibody, see Dilman p. 593, one of skill in the art would have a reasonable expectation of success of making the claimed monoclonal antibodies that have a cytotoxic activity in vitro against the cell line HepG2 in the presence of complement or peripheral blood mononuclear cells. Althought Filmus et al. teach that detection of GPC3 released in the serum, Filmus et al. also teach detection of GPC3 in the liver carcinoma tissue, see Tables 1 and 2 and Examples 4 and 5. Thus, given that both Filmus et al . and Midorikawa teach that GPC3 is expressed in hepatocellular carcinoma and given that Mast et al. teach that GPC 3 is expressed on the cell surface of HepG2 cells, one of skill in the art would have been motivated with a reasonable expectatin of success of making recombinant humanized monoclonal antibodies that has a cytotoxic activity in vitro against the cell line HepG2 in the presence of complement or peripheral blood mononuclear cells in view of Steplewski and Dillman who teach how to make humanized monoclonal antibodies that have ADCC or CDC activity in the presence of complement or peripheral blood mononuclear cells.

Double Patenting

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

4. Claims 9 and 23-29 provisionally rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 3, 6, 7, 16, 21, 22, 29, 32, 34, 38, 39, 41, 43-50 of copending Application No. 10/583,795. Although the conflicting claims are not identical, they are not patentably distinct from each other because the isolated recombinant, humanized monoclonal antibodies with a human kappa constant chain and a human gamma-1 H chain constant region claimed in claims 3, 6, 7, 16, 21, 22, 29, 32, 34, 38, 39, 41, 43-50 of copending Application No. 10/583,795 bind to epitope bind to epitopes in amino acids 375-580 of GPC3/SEQ ID NO:4 and have ADCC and CDC activity against HepG2 cells in vitro, and thus are a species of the claimed instantly claimed cytotoxic monoclonal antibodies. See Table on pages 21-24 Examples 6, 13-29, and Figs. 8, 12, 19 and 20.

This is a provisional obviousness-type double patenting rejection because the conflicting claims have not in fact been patented.

5. No claims allowed.

6. Any inquiry concerning this communication or earlier communications from the examiner should be directed to PETER J. REDDIG whose telephone number is (571)272-9031. The examiner can normally be reached on M-F 8:30 a.m.-5:00 p.m.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Larry Helms can be reached on (571) 272-083232. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Peter J Reddig/
Examiner, Art Unit 1642